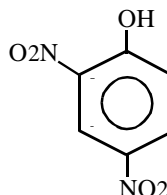


2,4-DINITROPHENOL

2,4-Dinitrophenol is a federal hazardous air pollutant and was identified as a toxic air contaminant in April 1993 under AB 2728.

CAS Registry Number: 51-28-5

Molecular Formula: $C_6H_4N_2O_5$



2,4-Dinitrophenol is a yellow, crystalline solid that has a sweet, musty odor. It sublimes when carefully heated and is volatile with steam. It is soluble in water (its crystalline sodium salts are also soluble in water), cold water (sparingly), ethyl acetate, acetone, chloroform, pyridine, carbon tetrachloride, toluene, alcohol, benzene, and aqueous alkaline solutions (Merck, 1989). It forms explosive salts with alkalis and ammonia, and emits toxic fumes of nitrogen oxides when heated to decomposition (Sax, 1989). It is incompatible with heavy metals and their compounds (Sittig, 1985).

Physical Properties of 2,4-Dinitrophenol

Synonyms: alpha-dinitrophenol; aldifen; DNP

Molecular Weight:	184.11
Melting Point:	112 - 114 °C
Vapor Density:	6.35 (air = 1)
Density/Specific Gravity:	1.683 at 24/4 °C (water = 1)
Vapor Pressure:	2.0×10^{-5} mm Hg at 25 °C
Log Octanol/Water Partition Coefficient:	1.5 - 1.54
Conversion Factor:	1 ppm = 7.53 mg/m ³

(HSDB, 1991; Merck, 1989; U.S. EPA, 1994a)

SOURCES AND EMISSIONS

A. Sources

Sources of 2,4-dinitrophenol include manufacturing plants, mines, foundries, metal, petroleum, and dye manufacturing plants which use 2,4-dinitrophenol. It is used as a wood preservative, indicator, reagent, chemical intermediate for production of azo dyes, and in the manufacturing of photographic developers (HSDB, 1991). 2,4-Dinitrophenol is also found in motor vehicle exhaust. It also may form as a result of photochemical reaction between benzene and nitrogen monoxide in polluted air (Howard, 1990).

2,4-Dinitrophenol was registered for use as a pesticide, however as of December 31, 1991, it is no longer registered for pesticidal use in California (DPR, 1996).

B. Emissions

No emissions of 2,4-dinitrophenol from stationary sources in California were reported, based on data obtained from the Air Toxics “Hot Spots” Program (AB 2588) (ARB, 1997b).

C. Natural Occurrence

No information about the natural occurrence of 2,4-dinitrophenol was found in the readily-available literature.

AMBIENT CONCENTRATIONS

No Air Resources Board data exist for ambient measurements of 2,4-dinitrophenol.

INDOOR SOURCES AND CONCENTRATIONS

No information about the indoor sources and concentrations of 2,4-dinitrophenol was found in the readily-available literature.

ATMOSPHERIC PERSISTENCE

In the atmosphere, 2,4-dinitrophenol may exist in the gaseous or particulate form. It may be removed by direct photolysis, by settling or washout in precipitation, or it may react in the gas-phase with photochemically-produced hydroxyl radicals (Howard, 1990). Based on the hydroxyl radical rate constant for 2-nitrophenol, the calculated half-life of 2-nitrophenol, due to reaction with hydroxyl radicals, is estimated to be 11 days. 2,4-Dinitrophenol is expected to be less reactive than 2-nitrophenol. 2,4-Dinitrophenol is therefore expected to be removed from the atmosphere by direct photolysis and/or wet and dry deposition (Atkinson, 1995).

AB 2588 RISK ASSESSMENT INFORMATION

2,4-Dinitrophenol emissions are not reported from stationary sources in California under the AB 2588 program. It is also not listed in the California Air Pollution Control Officers Association Air Toxics “Hot Spots” Program Revised 1992 Risk Assessment Guidelines as having health values (cancer or non-cancer) for use in risk assessments (CAPCOA, 1993).

HEALTH EFFECTS

Probable routes of human exposure to 2,4-dinitrophenol are inhalation and dermal contact (Howard, 1990).

Non-Cancer: 2,4-Dinitrophenol is a potent uncoupler of oxidative phosphorylation, and may cause methemoglobinemia. Acute exposure orally in humans has resulted in nausea, vomiting, sweating, dizziness, and headache. Chronic oral exposure in humans and animals has resulted in formation of cataracts and skin lesions and has caused effects on the bone marrow, central nervous system, and cardiovascular system (U.S. EPA, 1994a).

The United States Environmental Protection Agency (U.S. EPA) has determined that data are inadequate to set a Reference Concentration (RfC) for 2,4-dinitrophenol, but has established an oral Reference Dose (RfD) of 0.002 milligrams per kilogram per day based on cataract formation in humans. The U.S. EPA estimates that consumption of this dose or less, over a lifetime, would not likely result in the occurrence of chronic, non-cancer effects (U.S. EPA, 1994a).

No information is available on adverse reproductive or developmental effects of 2,4-dinitrophenol in humans. According to available animal studies, fetal growth inhibition, but no birth defects, were reported in the offspring of animals fed 2,4-dinitrophenol (U.S. EPA, 1994a).

Cancer: No information is available on the carcinogenic effects of 2,4-dinitrophenol in humans. Results from one oral study in mice indicated no tumor formation occurred after six months exposure, and in another study results indicated 2,4-dinitrophenol did not promote tumor development in mice. The International Agency for Research on Cancer and the U.S. EPA have not classified 2,4-dinitrophenol for potential human carcinogenicity (IARC, 1987a; U.S. EPA, 1994a).

